PAPERS AND ORIGINALS

Diet and heart: a postscript

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British Medical Journal, 1977, **2**, 1307-1314

Summary

During 1956-66, 337 healthy middle-aged men in London and south-east England participated in a seven-day individual weighed dietary survey. By the end of 1976, 45 of them had developed clinical coronary heart disease (CHD) which showed two main relationships with diet. Men with a high energy intake had a lower rate of disease than the rest, and, independently of this, so did men with a high intake of dietary fibre from cereals. Energy intake reflects physical activity, but the advantage of a diet high in cereal fibre cannot be explained; there was no evidence that the disease was associated with consumption of refined carbohydrates. Fewer cases of CHD developed among men with a relatively high ratio of poly-unsaturated to saturated fatty acids in their diet, but the difference was not statistically significant.

Introduction

At various times during 1956-66 the Medical Research Council's social medicine unit made dietary studies of 337 healthy working men aged 30-67 years. The men were representative samples of London and country staff from four of the big banks and of London Transport drivers and conductors. They represented 83% of men approached and eligible—that is, they had no history or clinical evidence of coronary heart disease, and none was on a prescribed diet. Of the remainder, 10% refused to participate and 7% produced incomplete records. As the sample of habitual diet we used a seven-day individual weighed record.[1 2] Half of the bank men completed two separate weeks of weighed surveys, and their results were averaged (table I). The studies were done mainly as part of a search for a practicable way of assessing individual diet that could be used in a large-scale prospective study of diet and coronary heart disease (CHD).[3-6] We failed to find a valid and simple enough method suitable for large-scale study, however, and eventually the project was abandoned.

Meanwhile, much information accumulated over the years about morbidity and mortality among the 337 men, from further surveys of the busmen at five[7] and 10 years,[6 8] from the personnel records of London Transport and the banks, and by correspondence with retired men. Furthermore, in accordance with the unit's routine all the men were "tagged" at the Registrar General's Office, and as any of them have died a copy of the notification has been sent to us. Up to the end of 1976, 45 of the men (13%) were known to have developed clinical CHD before the age of 70, 26 of whom (8%) had died (table I).

TABLE I—*Data on 337 men participating in seven-day individual weighed dietary survey during 1956-66*

		No
Participants in survey:		
Bank staff (clerks, cashiers, managers)		151
No completing second, separate week of survey	76	
Bus drivers		102
Bus conductors		84
No of cases of CHD up to end of 1976 (age 40-70)		45
No of deaths from CHD up to end of 1976 (age 40-70)		26
No of deaths from all causes up to end of 1976 (age 40-70)		51
No of men subjected to clinical examination		280 (270*)
No of them with CHD (age 40-70)	37 (35*)	

*Number on whom complete data available.

Present report

This report is concerned with diet and CHD. Information on the initial diet of the men is related to the 10-20 years of follow-up in terms of current notions on diet and the aetiology of CHD—the hypotheses or speculation on overeating, saturated fat and, more recently, refined carbohydrates and fibre deficiency. The information on morbidity from CHD may be considered to be reasonably complete till retirement of the men (or change of employer), and thereafter less so. Information on mortality will continue to be complete unless men emigrate. Antecedent data, however, are lacking in nine cases, known

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only through the Registrar General, so we cannot say which of them were "sudden deaths" in the first attack or at the end of first or later infarctions etc. Because of this, the small numbers, and the range of ages and occupations no attempt has been made to compare the modes of presentation of the disease. Incompleteness of information mainly affects the last 10 years of observation, and seven of the 15 cases in that period are drawn from death certificates alone. There is no reason to suppose that any of the deficiencies in data, or the inevitable variation in diagnosis, are systematically related to the men's diets. The total of 45 cases of CHD (table II) and the number of deaths were about what would be expected from general experience.[6-8]

TABLE II—*Main subsets of data*

Variable measured	No of men observed	Man-years of observation (age 40-70)	Cases of CHD (age 40-70)	Incidence of CHD per 1000 man-years
Diet, age and occupation, and height and weight	337	4601	45	9·78
Diet, smoking history, and clinical and biochemical variables	270	3585	35	9·76

To avoid importing bias the diagnoses made during the various surveys, or given on the sickness records and death certificates, have been accepted without question. Similarly, the dietary analyses done in 1956-66 were not reviewed; these used specially prepared food tables, for which we are indebted to the Ministry of Agriculture, Fisheries and Food (1956). We also drew on the manufacturers for some items—on Messrs Unilever, for example, for the fatty acid composition of the margarines being used. In those years, however, no estimate was made of the "fibre" content of the diets; indeed, satisfactory data for the dietary fibre in cereals have become available only recently.[9 10] This entailed a return to the original individual records, which we reanalysed in detail for the fibre content of the foods.

We then examined whether it was worth studying these men as a "population." Such seven-day individual weighed records are scarce; for a "cohort," however, the men were heterogeneous and small in number, though providing over 5000 man-years' experience in all, and prospective study had not been planned. The bank staff in three areas and all the busmen—280 of the men—had been examined clinically at around the date of the dietary survey, and complete data were available on 270 (table I). Their medical histories were therefore examined for standard risk factors for CHD. The results (see table III, for example) were encouraging: subsequent CHD was related to age and cigarette-smoking (table III (A)). Table III (B) shows the expected prediction also in relation to systolic blood pressure, plasma cholesterol concentration, and stature; some association was apparent with weight, though not with Quetelet's index or skinfold thickness. Since the men were divided into three equal groups, about the same number of cases should have occurred in each; instead they fell, 7, 13, and 16 by cholesterol concentration, etc.

Direct analyses were done on the reports at 40-70 years of age of clinical CHD among the 337 men against energy intake and many nutrients. There were no cases under 40 and only two over 70. Each man was counted only once, and the attack of CHD included was near enough a first attack to be called *incidence*; more accurately, we are dealing with the rate of *first known attack*. The following are the dietary items studied: energy; protein (animal, vegetable, and total); fats (animal, dairy, marine, and vegetable; polyunsaturated fatty acids (P), saturated fatty acids (S), and P:S ratio; total fat; and dietary cholesterol); carbohydrates (visible added sugar, total sugar; total carbohydrate); dietary fibre (from fruit, vegetables including potatoes, nuts, pulses; from cereals—whole grain, other, and total from cereal sources; total dietary fibre); alcohol; added salt (limited data). Wherever appropriate, these items were examined in terms of both total daily intake and intake per 1000 kcal (4·18 MJ) of the total diet.

Results

There were two main findings: they concerned energy intake and dietary fibre.

ENERGY INTAKE AND CHD

The coronary attack rate among the men was related to their energy intake but not in the way expected: high intake was associated with a low risk of CHD (table IV). Out of 112 men who were in the "high third" of the distribution for intake of energy at the initial survey, seven developed CHD; whereas of the 112 men in the low third, 23 developed the disease. The trend was evident through the 20 years of

TABLE III—*Incidence of clinical CHD during 1956-76 in relation to "risk factors" at time of initial survey (1956-66) in 337 men aged 30-67*

A				B											
Age at entry		Cigarette smoking		Remaining variables in thirds of distribution of men	Plasma cholesterol†		Systolic blood pressure†		Height§		Weight§		Index of weight for height‖		Skinfold thickness†	
No of men (years of age)	Cases of CHD*	No of men (cigarettes/ day)	Cases of CHD*		mmol/l‡	Cases of CHD*	mm Hg	Cases of CHD	cm‡	Cases of CHD	kg	Cases of CHD	kg/m²‡	Cases of CHD	mm, sum of three¶	Cases of CHD
77 (30–44)	7	89 (none)	6	Low third	3·0–5·6	7	100–128	7	151–171	22	46·8–67·5	20	15·9–22·5	15	12·2–27·1	12
118 (45–49)	16	80 (1–10)	5	Middle third	5·6–6·5	13	130–142	13	171–176	14	67·7–77·7	13	22·5–25·5	13	27·2–43·2	9
114 (50–59)	17	91 (11–20)	19	High third	6·5–8·6	16	144–218	17	177–190	8	78·0–106·4	11	25·5–33·3	16	43·3–91·9	16
28 (60–67)	5	21 (>20)	6													

*During 1956–76; 40–70 years of age.
†At initial survey (1956–66), 280 men underwent clinical examination (complete data available on 270)—that is, about 90 in each third. Of these, 37 (complete data available on 35) developed clinical CHD.
‡Overlap in values due to rounding.
§Height and weight were measured for 332 of the 337 men, 44 of whom developed CHD.
‖Quetelet's index: wt/(ht)².
¶Triceps + subscapular + suprailiac.
Conversion: SI to traditional units—Plasma cholesterol: 1 mmol/l ≈ 38·6 mg/100 ml.

TABLE IV—*Daily energy intake at time of dietary survey (1956-66) and incidence of clinical CHD during 1956-76 in 337 men*

Thirds of distribution of men*	Daily ranges of energy intake (kcal)†‡	Cases of CHD	Age-standardised incidences/ 1000 man-years of observation	Cases of CHD			CHD in each occupation			Total deaths from CHD
				In first 5 years	In second 5 years	In 10–20 years	Bank staff	Bus drivers	Bus conductors	
Low third (n = 112)	1860–2680	23	15·5	10	7	6	9	6	8	15
Middle third (n = 113)	2620–3070	15	9·7	6	2	7	7	5	3	5
High third (n = 112)	2840–4440	7	4·4	1	4	2	3	1	3	6

*Thirds composed of 112, 113, and 112 men respectively, classified according to thirds of energy intake in each occupation group; whence overlap of ranges.
†Total individual data divided by seven to give daily average; for men participating in two weeks of surveys, data divided by 14.
‡Conversion factor for kcal to MJ 0·00418; 1000 kcal = 4·18 MJ.

TABLE V—*Mean daily energy intake observed in future cases of CHD compared with average expected* on hypothesis that there is no association between energy intake and incidence of CHD*

Age at attack of CHD (years)	Bank staff			Bus drivers			Bus conductors			All occupations		
	No of cases	Energy intake (kcal)†		No of cases	Energy intake (kcal)		No of cases	Energy intake (kcal)		No of cases	Energy intake (kcal)	
		Observed	Expected		Observed	Expected		Observed	Expected		Observed	Expected
40–49	4	2769	3015	2	2918	2853				6	2819	2961
50–59	8	2514	2894	4	2808	2838	5	2515	2845	17	2583	2867
60–69	7	2725	2846	6	2458	2833	9	2718	2828	22	2649	2835
Total	19	2645	2902	12	2651	2838	14	2646	2834	45	2647‡	2864‡

*See text.
†1000 kcal = 4·18 MJ.
‡$t = -3{\cdot}36$; $P < 0{\cdot}001$. Observed and expected standard deviations (within age and occupation groups) were 387 and 434 kcal respectively.

TABLE VI—*Daily energy intake and physique at time of initial survey (1956-66)*

Thirds of distribution of men*	Daily ranges of energy intake (kcal)*	Mean height (cm)	Mean body weight (kg)	Mean Quetelet index (kg/m^2)	Mean skinfold thickness† (mm)	Mean energy intake (kcal) per kg body weight		
						Bank staff	Bus drivers	Bus conductors
Low third	1860–2680	172	68·5	23	34·3	34	34	40
Middle third	2620–3070	174	73·4	24	40·0	37	38	43
High third	2840–4440	174	75·6	25	40·2	42	45	48

*See table IV.
†See table III.

observation, though clearer in the first five, with 10, six, and one case respectively in the thirds of the men, and was seen in each occupation —white collar, skilled, and part-skilled.

The men were grouped into thirds according to energy intake within occupations so as not to bias the results through occupational differences; but there was still a possibility of more subtle biases from different durations of observation—that is, experience—or from variations in age structure in the energy comparison groups (energy intake declines and incidence of CHD rises with age). Thus *age-standardised* incidences based also on calculations of *man-years of observation* were used (see Appendix for method): these showed a somewhat stronger trend (table IV).

The significance of the inverse association between energy intake and CHD was assessed from the more efficient analysis shown in table V. In table V the observed mean energy intake of men in each occupation who suffered an attack during each of three bands of age is compared with the expected value if energy intake were unrelated to the risk of CHD. These expected values are calculated from the energy intakes of all men in each occupation observed during each age band; differences in the length of time observed are allowed for by weighting. In future cases the energy intake observed was lower than expected in all except drivers aged 40-49. In the marginal column and row of table V the expected values take account also of the distribution of cases between cells. The table thus allows examination of the findings (*a*) within occupations, adjusted for age; (*b*) within age bands, adjusted for occupation; and (*c*) overall, adjusted for both age and occupation. This overall comparison confirms the association ($P < 0{\cdot}001$) between low energy intake and high risk of CHD shown in table IV. Details of the calculations in table V are given in the Appendix.

Pattern of diet

There was little *qualitative* difference in the patterns of diet between the three energy-intake groups (fig 1). Thus in the thirds of the men, saturated fats, for example, constituted 21·5%, 21·2%, and 21·4% of total energy intake.

Physique

Analysis of physique and CHD yielded nothing of particular relevance. Men with a low energy intake were lighter and thinner on average than the rest, and somewhat shorter (table VI). Nevertheless, there was no indication that thinness itself was predictive of CHD, thin men apparently being at high risk only if in the low-energy-intake group. On the other hand, the skinfold thicknesses of men in the middle and high intake groups were unremarkable, with little serious obesity in either group.

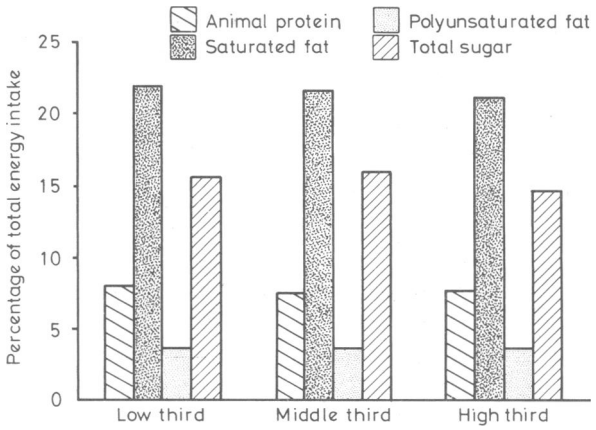

FIG 1—Proportions of nutrients in diets of men in thirds of distribution for energy intake.
Cereal-fibre intake (g/1000 kcal (4·18 MJ)) in thirds, from low to high, averaged 2·76, 3·01, and 3·21 respectively.

As previously reported, other factors being equal, short men are at greater risk of suffering CHD than tall, and the shorter men are likely to eat less. The two "effects," however, seem to be independent. Thus the mean daily energy intake in future cases was lower than expected in each third of the height distribution (table VII). This is the only nod we can make at present to "familial" factors.

TABLE VII—*Daily energy intakes in cases of CHD grouped in thirds according to height*

Stature of men	Cases of CHD	Mean daily energy intake (kcal*)	
		Observed in cases	Expected†
Short third	22	2632	2790
Middle third	14	2670	2898
Tall third	8	2681	2826

*1000 kcal = 4·18 MJ.
†Adjusted for age, occupation, and man-years of observation.

Smoking

Cigarette-smoking predicts later disease, and there were rather more who smoked among the men with a low energy intake. But again the two effects were distinct, the mean energy intake of men with CHD being less than expected in each smoking category (table VIII).

TABLE VIII—*Daily energy intakes in cases of CHD and cigarette smoking*

No of cigarettes/day	Cases of CHD	Mean daily energy intake (kcal*)	
		Observed in cases	Expected
0	6	2766	2891
1–10	5	2533	2752
11–20	19	2641	2841
>20	6	2706	2844

*1000 kcal = 4·18 MJ.

FIBRE DEFICIENCY

The hypothesis, as variously described,[12 15] states that depletion of fibre in the diet—from high consumption of sugar, fats, meat, and dairy foods; and because we eat fewer cereals, which in any case are highly refined—is a factor in the historic increase and modern prevalence of CHD. These indeed represent epochal changes in Western diet and community health.[16 18]

In the present data the incidence of CHD was related to *total* fibre in the diet (table IX): low intake with more disease, and high intake with relative freedom from it. On further analysis, however, the association was found only with fibre from cereals; the other and equally large part of dietary fibre—namely, that derived from fruit or vegetables, pulses, and nuts—was not related to CHD. The association with cereal-fibre intake (table X) was apparent through the 20 years of experience and for 2 of the 3 occupations. The age-standardised attack rate again showed a somewhat wider range than the numbers of cases.

TABLE IX—*Daily dietary fibre intake at time of initial survey (1956-66) and incidence of CHD during 1956-76 in 337 men*

Thirds of distribution of men*	Total dietary fibre		Fibre from fruit, vegetables, pulses, and nuts		Fibre from cereals	
	g/man/day	Cases of CHD	g/man/day	Cases of CHD	g man/day	Cases of CHD
Low third	5·6–15·4	22	0·6–6·9	14	2·0–7·1	25
Middle third	13·8–19·0	16	6·1–9·0	18	6·4–9·7	15
High third	16·9–56·1	7	8·3–26·5	13	8·4–34·2	5

*Thirds composed of 112, 113, and 112 men respectively, classified according to thirds of dietary fibre intake in each occupation.

Men with a relatively low energy intake ate less of virtually everything, including cereals, and men in the high third for energy intake tended to eat a lot in general, including cereals. To disentangle the two factors, table XI shows cereal-fibre intake in g/1000 kcal (4·18 MJ). The finding is now consistent for the three occupations. In seven of the eight cells future cases show a lower than expected intake of dietary fibre from cereals. Overall, after adjusting for age, for occupation, and for length of follow-up, men who developed CHD had a mean intake of 2·42 g of cereal fibre per 1000 kcal, compared with 2·84 g expected on the null hypothesis ($P < 0·005$). Or, as a simpler way of looking at it, without adjustments: in the 45 future cases a daily average intake of 6·7 g of dietary fibre from cereals was recorded, whereas the 292 men who did not develop clinical CHD during the period had a daily average intake of 8·9 g. The *t* values suggest that intakes of cereal fibre and energy were of about the same importance for predicting CHD in these men. (Such more elaborate analysis of the rest of dietary fibre yielded as random results as shown in the centre column of table IX.)

Pattern of diet

Table XII and fig 2 give the sources of the cereal fibre eaten. The importance of brown, including some wholemeal, bread and of breakfast cereals is evident. No bran was taken as such (this was 1956-66), and little "high-bran" breakfast cereals.

Figure 3 illustrates the main features of the diet as a whole of the men by thirds of cereal-fibre intake and is quite uniform. The composition of the diet among men in the high third of intake shown in fig 3, or for that matter in fig 2, does not impress as being particularly "health conscious."

Smoking

There was some clustering again in respect of smoking, more of the men who ate little cereal fibre being cigarette-smokers, and fewer of those eating a lot. But again the two effects seemed to be independent (table XIII).

TABLE X—*Intake of dietary fibre from cereals at time of initial survey (1956-66) and incidence of CHD during 1956-76 in 337 men*

Thirds of distribution of men	Daily ranges of cereal-fibre intake (g)	Cases of CHD	Age-standardised incidences/1000 man-years of observation	Cases of CHD			CHD in each occupation			Total deaths from CHD
				In first 5 years	In second 5 years	In 10–20 years	Bank staff	Bus drivers	Bus conductors	
Low third	2·0– 7·1	25	17·1	10	7	8	12	4	9	16
Middle third	6·4– 9·7	15	9·8	6	4	5	5	5	5	6
High third	8·4–34·2	5	3·1	1	2	2	2	3	0	4

TABLE XI—*Mean daily dietary cereal-fibre intake (g/1000 kcal (4·18 MJ))* observed in future cases of CHD compared with average expected on hypothesis that there is no association between cereal-fibre intake and incidence of CHD*

Age at attack of CHD (years)	Bank staff			Bus drivers			Bus conductors			All occupations		
	No of cases	Cereal-fibre intake (g/1000 kcal)		No of cases	Cereal-fibre intake (g 1000 kcal)		No of cases	Cereal-fibre intake (g 1000 kcal)		No of cases	Cereal-fibre intake (g/1000 kcal)	
		Observed	Expected		Observed	Expected		Observed	Expected		Observed	Expected
40–49	4	2·63	2·98	2	2·80	2·71				6	2·68	2·89
50–59	8	2·15	3·01	4	2·03	2·61	5	2·58	2·79	17	2·24	2·84
60–69	7	2·73	3·04	6	2·55	2·77	9	2·29	2·68	22	2·49	2·82
Total	19	2·45	3·01	12	2·40	2·71	14	2·39	2·72	45	2·42†	2·84†

*This index was only slightly correlated with energy intake within occupations: $r = +0·17$. Owing to long-tailed distribution—lowest values were about 1 g, and highest 20 men had intakes of 5·0–9·4 g/1000 kcal—calculations were performed on logarithmic scale and transformed back to original scale for clearer presentation. Means are therefore geometric instead of arithmetic.

†$t = -3·26$; $P < 0·005$. Standard deviation, calculated on log scale, may be interpreted as coefficient of variation. Observed and expected values (within age and occupation groups) were 29·7% and 34·1% respectively. Observed mean differed from expected mean by 14·8%.

TABLE XII—*Sources of dietary fibre from cereals at time of initial survey (1956-66) in 337 men*

Thirds of distribution of men	Daily range of cereal-fibre intake (g)	Sources: mean intake in oz (g) week				
		White bread	Brown bread*	Biscuits, cakes, and pastry†	Breakfast cereals	Porridge
Low third	2·0–7·1	30 (850)	0·9 (25·5)	20 (567)	0·3 (8·5)	2·7 (77)
Middle third	6·4–9·7	40 (1134)	2·6 (73·7)	28 (794)	1·4 (40)	5·4 (153)
High third	8·4–34·2	38 (1077)	11·1 (315)	32 (907)	4·0 (113)	5·7 (162)

*Including some wholemeal bread.
†Including a little crispbreads.
Other sources—for example, soups—too small to estimate.

TABLE XIII—*Daily dietary fibre intake from cereals (g/1000 kcal) in cases of CHD and cigarette smoking*

No of cigarettes day	Mean daily cereal-fibre intake	
	Observed in cases	Expected
0	2·75	3·11
1–10	2·56	2·72
11–20	2·34	2·61
>20	1·90	2·28

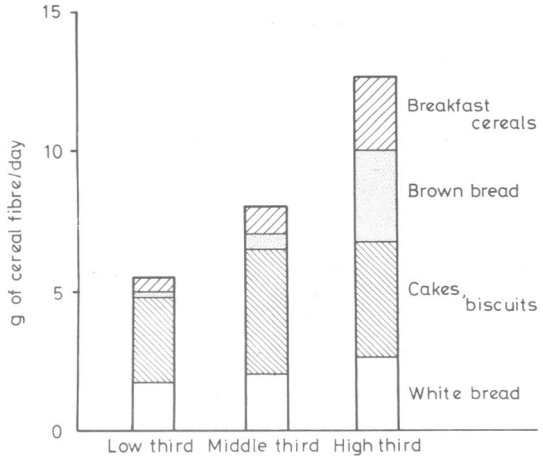

FIG 2—Sources of fibre among men in thirds of distribution for cereal-fibre intake: contribution of foods in g of cereal fibre per day. Total cereal-fibre in thirds, from low to high, averaged 5·3, 8·0, and 12·5 g, respectively.

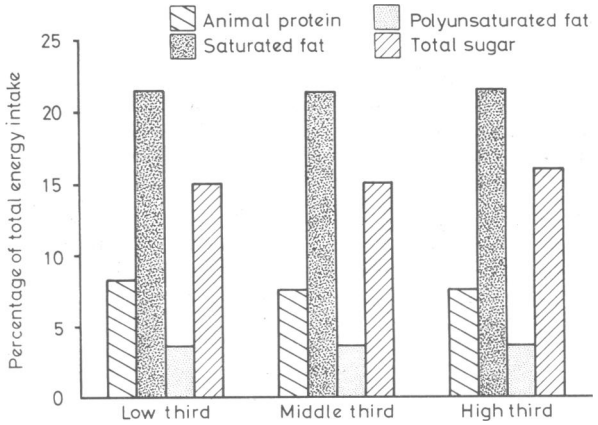

FIG 3—Patterns of diet of men in thirds of distribution for cereal-fibre intake: contribution of several nutrients to energy intake. Means of total kcal in thirds, from low to high, were 2558, 2868, and 3118 respectively. (1000 kcal = 4·18 MJ.)

We hope to make a return visit to the surviving men to bring the information on their health and dietary habits up to date, and will then also inquire about bowel function and abdominal symptoms.

Refined carbohydrates: sugar

There was no sign of a relation between the consumption of sucrose[14 19] and CHD. When the men were classified in thirds of the distribution for their total sugar intake (which ranged from 10 to 260 g daily) the numbers of cases fell 15, 19, and 11 from low to high. In g of sugar eaten per 1000 kcal the cases were distributed 17, 12, and 16 respectively. The 59 men taking less than 10% of total energy from sugar contributed eight cases, and the 52 taking 20% or more seven cases. Analysis, as in tables V and XI, shows the observed consumption of sugar in cases to be more than expected in three of the cells and less in five. The mean daily intake among the 45 future cases was 110 g compared with 114 g in the rest of the men.

No systematic relation was apparent between sugar eaten and cereal-fibre intake, whether estimated in g or as g/1000 kcal, the correlation coefficients varying from −0·03 to +0·13.

Straight tabulations of coronary cases against *white bread* eaten showed no trend. Thus our data do not support the hypothesis that the incidence of CHD is related directly to intake of refined carbohydrates.

DIETARY FATS AND CHD

Today's leading hypothesis on aetiology relates the incidence of CHD, through serum cholesterol concentrations, to the fat in the diet, and more particularly to the saturated fatty-acid content, or the ratio of polyunsaturated to saturated fatty acids (P:S ratio). Diets high in total fat, it is postulated, rich in saturated fat, or with a low P:S ratio are associated with a high incidence.[20–22] The dietary data were therefore analysed in detail for (*a*) *grammes of fat eaten*; (*b*) conventionally, the *proportion* these fats contributed to total energy intake; and (*c*) the P:S ratio. In view of the dominance of energy in the present data it would not be instructive to present (*a*); but table XIV, for example, is quite uninteresting.

TABLE XIV—*Proportion of total energy derived from fats at time of initial survey (1956-66) and incidence of CHD during 1956-76 in 337 men*

Energy from fats (% of total dietary energy) in thirds of distribution of men*	Total fat		Animal fat		Dairy fat		Marine and vegetable fats and oils	
	% Range of energy	Cases of CHD	% Range of energy	Cases of CHD	% Range of energy	Cases of CHD	% Range of energy	Cases of CHD
Low third	30–39	18	5·2–14	18	3·0–14	13	1·2–9·5	19
Middle third	38–43	10	12–18	13	11–17	14	8·5–12	15
High third	41–56	17	15–27	14	16–30	18	12–31	11

*As in previous tables.

The ratio of polyunsaturated to saturated fatty acids in the diets was small throughout, ranging from only 0·09 to 0·28; and the highest ratio was smaller than is often regarded as optimal.[20 21] Nevertheless, there was a significant association ($P < 0·05$) with CHD as postulated in the first five years of follow-up (table XV). The suggestion in table XV seems to be that men with a relatively high P:S ratio are at an advantage compared with the rest. Analysis of the data as in tables V and XI shows the observed means of the P:S ratio for the cases lower than would be expected in seven of the eight cells, in itself a singular enough finding, but the overall difference, 0·151 *v* 0·158, does not reach the 10% level of significance.

Analysis of dietary cholesterol against coronary events showed no association at all.

BLOOD LIPIDS

A massive correlation exercise was carried out on the data we have on 270 of the 280 men who were examined clinically for: plasma cholesterol (concentration); α- and β-lipoprotein cholesterol (concentration and percentage of total cholesterol); and Svedberg flotation units (0-12, 12-20, 20-100, and 100-400; 20-400 units indicates very low-density pre-β-lipoproteins).[3 7] All blood samples were "casual," non-fasting. Correlation coefficients were calculated between individual lipid values and all the dietary variables, firstly, for all the men, then standardised by occupation. The results throughout were, as expected,[3] little or nothing. Evidently in these data the association between energy intake or cereal-fibre intake or the ratio of P:S in the diet and CHD is not mediated or explained in any simple way through plasma cholesterol concentrations. Very few of the hundreds of correlation coefficients reached 0·2; later we shall study "cut-off" points etc.

BLOOD PRESSURE

No association was found between blood pressure and the nutrient factors studied.

SUMMING UP

Table XVI, after tables V and XI, summarises the six variables that on single-factor analysis (adjusted for age, occupation, and length of observation) significantly predicted CHD. Diastolic pressure was not as predictive as systolic. Weight, Quetelet's index, and skinfold thicknesses did not approach 10% significance on such analysis. Nor, with and without taking account of energy intake, did any other of the dietary items that we have not specifically discussed.

MULTIPLE FACTORS

Simple cross-tabulation suggests that the six factors contribute effects that are largely independent of each other; but, to be more confident of this, they have to be studied together. Variables like weight and diastolic blood pressure that there is reason to suppose from other studies may be predictive have again to be included; and, of course, age and occupation. Since this is primarily a study of diet and CHD, all the other dietary variables that on direct analysis showed nothing of interest have to be reintroduced into this multivariate exercise: to check, for example, that it is indeed energy intake that matters, and not some combination of the nutrients that make up total energy, and to test again for possible associations of CHD with "other" dietary fibre and with the fats in the diet. By such an approach it is conceivable also that some subtler relationships may be disclosed.

Multiple regression analyses were done. The results, which will be fully documented later, *firstly*, confirmed the importance and independence of the three behavioural factors (energy intake, dietary fibre from cereals, and cigarette-smoking) included in table XVI; *secondly*, raised none of the other "standard" variables, like weight and skinfold thickness, anywhere near significance; and, *thirdly*, suggested also that animal protein may somehow be involved: this emerges as negatively and significantly correlated with CHD. There is no sign of any such effect in the straight, single-factor analysis, whether or not adjusted for age, occupation, and length of follow-up. The cases in the thirds of the men, from low to high, in g of animal protein/1000 kcal fall 14, 19, and 12; and since its effect in the multiple regression is to enhance the size and significance of the *t* values for energy and cereal fibre, we are letting it rest for the moment. Later we shall study food sources of protein as well. When a multitude of correlation coefficients are calculated it must be expected that something statistically "significant" will turn up, which may or may not be of biological significance; an abiding dilemma of multivariate analysis.

Other vascular disease

Only 17 cases of cerebrovascular, peripheral vascular, and other vascular diseases were recorded.

TABLE XV—*Ratio of polyunsaturated (P) to saturated (S) fatty acids at time of initial survey (1956-66) and incidence of CHD during 1956-76 in 337 men*

Thirds of distribution of men*	Ranges of P:S ratios	Cases of CHD	Age-standardised incidences 1000 man-years of observation	Cases of CHD			CHD in each occupation			Total deaths from CHD
				In first 5 years	In second 5 years	In 10–20 years	Bank staff	Bus drivers	Bus conductors	
Low third	0·09–0·15	20	13·9	9	4	7	10	4	6	11
Middle third	0·13–0·18	18	11·7	7	5	6	6	5	7	10
High third	0·16–0·28	7	4·3	1	4	2	3	3	1	5

*Thirds composed of 112, 113, and 112 men respectively, classified according to thirds of P:S ratio in each occupation.

TABLE XVI—*Observed mean values of predictive variables in future cases of CHD compared with expected means on null hypothesis*

Variable	Bank staff		Bus drivers		Bus conductors		All occupations		
	Observed	Expected†	Observed	Expected†	Observed	Expected†	Observed	Expected‡	*t* value§
kcal/day*	2645	2902	2651	2838	2646	2834	2647	2864	−3·36 (P<0·001)
Cereal fibre (g/1000 kcal)	2·45	3·01	2·40	2·71	2·39	2·72	2·42	2·84	−3·26 (P<0·005)
No of cigarettes/day	15·0	5·9	15·6	9·7	14·9	11·5	15·1	9·4	+4·09 (P<0·0001)
Plasma cholesterol (mmol/l)	6·52	6·18	6·81	6·10	6·14	5·93	6·46	6·05	+2·34 (P<0·02)
Systolic BP (mm Hg)	140	130	145	142	145	141	144	138	+1·65 (P<0·10)
Height (cm)	171·1	177·0	171·8	172·2	166·2	169·2	169·7	173·2	−4·22 (P<0·0001)

*1000 kcal = 4·18 MJ.
†Expected means for each occupation take age structures into account.
‡Expected means for all occupations are adjusted for age and occupation.
§*t* Value is difference between observed and expected means, divided by standard error.
‖Analysis carried out on log scale.
Conversion: SI to traditional units—Plasma cholesterol: 1 mmol/l ≈ 38·6 mg/100 ml.

Other causes of death under age 70

Remarkably few deaths occurred under the age of 70—13 from malignant disease, and 12 from other causes. The small numbers may reflect that the sample was of men at work, with some other exclusions, and not a general population sample. In any event, numbers preclude analysis at present.

Discussion

Our main interest is in the connections between the mode of life—communal and individual behaviour—and health. Unequivocal results alone will be considered: others may or may not be due to small numbers, insensitivity in the methods used, and misclassification. Thus among the 76 bank men who completed two separate weeks of weighing, at intervals of one month to one year, to give us an idea of how typical a picture of habitual diet the seven-day individual weighing provided, the correlation between intake of energy in the first and second weeks was +0·73, for cereal fibre +0·8, for sugar intake +0·87, and for total fats only +0·66. The error in estimating the fat content of some important foods—for example, meat—from average values of nutrients given in food composition tables is apt to be serious. These are staple problems of dietary assessment in free-living people.

With regard to the observation on *energy*, there are hints of it in previous work,[23-26] though such are the general preconceptions (which we shared), that the suggestion of an inverse relation between energy intake and coronary disease has been overlooked. In seeking to understand the present finding, obesity plainly is irrelevant: there was no suggestion of it in the low-energy-intake men, and little serious suggestion in the high; reports of weight change in the initial survey and on follow-up were similar in the three energy-intake groups. The main observation, too, was unaffected on standardising for body size. Personal differences in basal metabolism, in energy expenditure for maintenance of routine bodily functions, and in the efficient use of energy will all be involved in the individual values of the men in the energy-intake groups. We cannot say how much; casual pulse rate, recorded at the initial clinical examination, was similar in the three groups, averaging 71, 73, and 72/min, for example, in the bank men. High cereal-fibre intake, which is associated with high energy intake and may result in increased loss of energy in the stools,[27] could also be a factor. And at the extremes, anyhow, the differences that were found, amounting to hundreds of kcal daily in energy turnover and output, would also reflect in some or large measure the habitual physical activity of the men.

A little direct evidence on the exercise taken by the men can be adduced, again serendipitously. Physical activity in leisure time had been recorded for the first sample of the bus men,[7] and this was analysed "blind," in respect of both occupation and diet, by a colleague. The results are interesting (table XVII):

TABLE XVII—*Analysis of activity of a sample of bus drivers in leisure time*

Thirds of distribution of men for energy intake	Physical activity in leisure time		
	Little	Moderate	Much
Low third	16	9	4
Middle third	9	12	3
High third	5	10	9

energy intake appeared to increase with physical activity. The conductors, with similar numbers, showed no such trend, nearly all of them being in the "moderate" group; their jobs were, of course, physically more demanding and provided a greater potential range of activity. Table VI shows that in each of the thirds for energy intake the conductors had a higher expenditure of energy per kg body weight. The data were less suitable for analysis of *vigorous exercise*, as now defined,[28] but a trend again was evident, with one, five, and six instances of CHD in the low-, middle-, and high-energy-intake groups of drivers, and this time there was a hint of it also in the conductors, with three, four, and seven instances respectively. In a previous study of male executive-grade office workers in the Civil Service there were significant correlations between estimates of physical activity in leisure time and total energy intake, measured by the same methods as in this study.[29]

Another possibility has to be considered—namely, that the high-energy-intake men enjoyed also a superior diet *qualitatively*, because, eating more in general, there will be less risk of absolute deficiencies; and that the small eaters may also have taken an inferior diet, carrying a risk of some deficiency. Thus it was noted (fig 1) that the low-energy-intake men had a little lower intake of cereal fibre, if not of the other nutrients studied. We have not yet pursued this notion in mineral, vitamin, and trace-element analysis. There was nothing glaringly wrong in the varied if not ample diets of many of the men with a low energy intake.

The finding on *cereal fibre* illustrates one of the uses of epidemiology—in exploration. If anything, what little is now known from the laboratory suggests that fibre from fruit, vegetables, and pulses are more likely to be related to CHD because of the experimental connection between (large) doses of the pectin and guar gum, for example, found in them, and plasma cholesterol concentrations.[30 31] There was no confirmation of any of this, however, in our data. The physical and chemical properties of cereal and fruit and vegetable fibre differ substantially,[9 32 33] so different physiological effects are to be expected. And among cereals, not only does wholemeal bread contain three times, and brown bread twice, as much dietary fibre as white bread (8·5, 5·1, and 2·7 g/100 g respectively) but the fibre in white and brown bread also differs.[9] Whole grains carry polyunsaturated fat (linoleate) in the wheat germ, though the quantity even in the 20 men recording a large intake of fibre (table XI) was too small to affect the overall P:S ratio of their diet—because of the milk that was added to porridge and breakfast cereals. The linoleate is only one possibility that cereal fibre may be the vehicle of other effectual nutrients.[34] So, meanwhile, no mechanisms can be postulated for the present observation (which could be tested fairly quickly); and since we ourselves are only now and painfully learning about "fibre," we shall not speculate. Historically, the observation makes sense: *if* dietary fibre is related to the "modern epidemic" of CHD, the link should be with the fibre in cereals. It is the intake of that which has fallen; potatoes apart, consumption of fruit and vegetables has if anything increased.[16 17 35] No evidence was found that refined carbohydrates were themselves involved. To those who wilt at the possibility of yet another behavioural risk factor for CHD it may be said that what is presently known by no means accounts for the occurrence of heart attack and immunity from it; that this one would be easier to put right than some; and, as in so much of today's health and prevention, makes good sense in itself.[6]

Meanwhile, a pattern of healthy living may have been identified: high energy intake and expenditure, high intake of cereal fibre, no cigarettes, with relatively little proneness to heart attack; and another behaviour pattern, of low energy intake and physical inactivity, low intake of cereal fibre, smoking cigarettes—carrying a relatively high risk. Health consciousness and personality would be worth studying among such men in the different conditions of today, together with the obvious biochemical and metabolic reactions for a start.

We acknowledge our gratitude to colleagues and friends who have collaborated in this study over the years: to the bank staff, the drivers and conductors, and their wives; the staff controllers of Barclays, Midland, National Provincial, and Westminster Banks Ltd; the chief depot inspectors of the bus garages; the chief medical officer of London Transport, Dr P A B Raffle; Drs A Kagan, T W Meade, and D C Pattison, former colleagues in the social medicine unit, for making

the medical examinations and surveys; Dr G L Mills, Courtauld Institute of Biochemistry, Middlesex Hospital, and Professor T R E Pilkington, St George's Hospital, for the lipid analyses; the General Register Office—OPCS; Ms C Murland and M Bramwell for some of the dietary surveys; Mrs P Parker and Mrs E Plumridge for many hours of calculation before the days of computers; Mr J Woods for many hours of skilled computer-programming; Dr S P W Chave for classifying physical activity; Dr C T M Davies, Dr J A Heady, Mrs G Mann, and Dr D A T Southgate for personal advice; friends for critical reading of the drafts; the secretarial staff for much patient typing; and the many other helpers we are unable to name.

Appendix

STATISTICAL METHODS FOR LONG AND VARIED FOLLOW-UP PERIODS

The prolonged and very variable follow-up times in this study necessitated the use of statistical methods based on life tables. All the methods used entailed partitioning the total time for which each man was observed into the times spent in each of the three age bands—namely, 40-49, 50-59, and 60-69 years. The attack of CHD similarly was classified according to the age at which it occurred. The analyses were repeated by five-year age bands, with no appreciable effect on the results.

Age-standardised incidence rates were calculated from the number of "first" attacks occurring during each age band in each of the groups being compared—thirds for energy intake (table IV), thirds for cereal-fibre intake (table X), thirds for P:S ratio (table XV)—together with the corresponding man-years of "exposure to risk." From such tabulations standardised rates were calculated by the indirect method proposed by Mantel and Stark.[36]

"Expected" mean values in men who suffered an attack of CHD (tables V, XI, and XVI) similarly take account of the variable durations of exposure to risk. Let x_{ij} represent the value of the variable for the i^{th} man in the j^{th} occupation, and let t_{ijk} represent the time spent by that man in the k^{th} age band. If the variable x has no relation with risk of CHD its "expected" mean value in the men in the j^{th} occupation, who suffered an attack during the k^{th} age group, is given by

$$\bar{x}_{jk} = \sum_i x_{ij}t_{ijk} \Big/ \sum_i t_{ijk}.$$

This is a weighted average, taking the exposure times as weights. The expected mean for men in the j^{th} occupation who suffered an attack of CHD in any of the three age bands is corrected for the age distribution of attacks, and is given by

$$\bar{x}_j = \sum_k d_{jk}\,\bar{x}_{jk} \Big/ \sum_k d_{jk}.$$

This is again a simple weighted average, over age, of the expected means x_{jk}, now taking the number of attacks occurring in each cell d_{jk} as weights. Similarly the expected mean for men of all occupations who suffered attacks in the k^{th} age group is corrected for the distribution of attacks between occupations, and is given by

$$\bar{x}_k = \sum_j d_{jk}\,\bar{x}_{jk} \Big/ \sum_j d_{jk}.$$

The overall mean value for all men who suffered an attack, on the null hypothesis of "no relationship with risk," and correcting for the distribution of attacks by age and occupation, is given by

$$\bar{x} = \sum_{jk} d_{jk}\,\bar{x}_{jk} \Big/ \sum_{jk} d_{jk}.$$

The significance of the difference between the observed mean for cases and this "expected" value is assessed by dividing the difference by its standard error. The formula for calculating the standard error is

$$\frac{1}{\sum_{jk} d_{jk}} \sqrt{\sum_{jk} d_{jk} \left| \frac{\sum_i t_{ijk}\, x^2_{ij} - (\bar{x}_{jk})^2}{\sum_i t_{ijk}} \right|}.$$

The resultant t statistic is asymptotically distributed according to the standard normal distribution.

Multiple regression analysis is not reported in detail but was based on similar considerations. The variability of exposure times precluded the use of the familiar logistic regression and, instead, a method based on the regression model for life tables, proposed by Cox, was used.[37]

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(*Accepted 27 September 1977*)